Case Discussion in Urology

Case Based Discussion on Benign Prostatic Hyperplasia (BPH)

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Benign Prostatic Hyperplasia (BPH) has been historically used to define a disease characterized by symptoms of urinary dysfunction as a consequence of an obstacle to micturition caused by an enlarged prostate. Recently, BPH has been referred to a histologic diagnosis characterized by the proliferation of smooth muscle and epithelial cells within the transition zone of prostate. LUTS (lower urinary tract symptoms) are considered as a clinical manifestation with multifactorial pathophysiology including prostatic and non-prostatic etiology. The proper diagnostic assessment of men with LUTS represents a major issue in the everyday clinical practice. The workup should aim at evaluating severity of symptoms and their impact on quality of life so that severity of disease, response to therapy and risk of disease progression could be assessed. IPSS (international prostate symptom score) is currently considered the international standard tool to investigate LUTS severity. Based on the IPSS, the treatment of BPH can differ for each patient. Here, we present some case-based discussion of BPH at each stage of disease and how the treatment of each case differs from each other case.

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Key words **: BPH, LUTS, IPSS.**

Benign Prostatic Hyperplasia (BPH) is a pathologic process characterized by an increased number of epithelial and stromal cells in the periurethral area of prostate. Normal prostate consists of 70% glandular and 30% stromal component. Prostate volume of approximately 20 ml may be regarded as normal.

Androgens not only are required for normal cell proliferation and differentiation in the prostate but also actively inhibit cell death. Etiological factors include high levels of prostatic dihydrotestosterone (DHT) & androgen receptors (AR), increased levels FGF-1, FGF-2, FGF-7, VEGF, IGF, first-degree male relative of surgically treated BPH with autosomal dominant inheritance.

BPH result in anatomic Bladder Outlet Obstruction (BOO), through two distinct mechanisms: first, an increase in prostate volume, termed the static component. Second, an increase in stromal smooth muscle tone, termed the dynamic component. Notably, two factors complicate the natural history and clinical presentation of BPH, BOO and LUTS; first, prostate volume does not linearly correlate with the severity of BOO or LUTS. Second, progressive BPH and BOO can lead to primary bladder dysfunction, which in turn can exacerbate the severity of LUTS independently of BOO.

Editor's Comment :

- Clinical examination of patient, assessment of symptom severity and risk of disease progression and associated comorbidities are utmost essential before determining the definitive treatment of BPH.

Approximately 50% of all men >50 years, with BPH will eventually develop Lower Urinary Tract Symptoms (LUTS). Nocturia is most common symptom followed by frequency and urgency. The International Prostate Symptom Score (IPSS) is recommended as the symptom scoring instrument to be used for the assessment of symptom severity in men presenting with LUTS.

Case 1 :

A 60-year-male presented with increased frequency, narrow stream of urine and sensation of incomplete evacuation of urine for 2 years.

History of Poor urinary stream (2), Straining during micturition (2), Intermittency (1), Incomplete urinary bladder emptying (2), Frequency (4), Urgency (2), Nocturia (4); IPSS 17/35. There was no history of hematuria, urethral discharge, constipation, low back pain or limb weakness.

Surgical history and family history were not significant. External genitalia – normal, Hernial sites – no cough impulse. DRE- peri-anal skin – normal,

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anal tone – normal, prostate-grade II, firm, smooth, non-nodular, non-tender, median sulcus palpable, rectal mucosa freely mobile.

On further investigation ultrasound examination showed no evidence of bilateral hydroureteronephrosis, urinary bladder wall thickened, prostate – 42 cc without median lobe enlargement, PVR- 94ml. No evidence of UTI and hematuria on urinalysis. Serum urea / creatinine was 22/0.8 and S. PSA- 2.4 ng/ml. Uroflowmetry showed – Voiding volume 260ml, Q max – 13 ml/sec.

As the patient presented with moderate IPSS, PVR <300 ml and no absolute indication for surgical intervention; the patient was planned for medical therapy for BPH.

Alpha adrenergic blockers- the rationale for its use is based on the BPH mediated BOO, which is due by α_1 adrenergic receptor associated with prostatic smooth muscle causing dynamic obstruction. ADRs includes postural hypotension, 1st dose hypotension, dizziness, fatigue, headache, asthenia, nausea. Tamsulosin (0.4 mg OD HS) is currently the most widely employed α_1 blocker (higher affinity for α_{1a} and α_{1d}) for BPH as it has minimum effect on BP, no dose titration needed; common side effect is retrograde ejaculation. Slow release alfuzosin allows for once daily dosing regimen (10 mg once a day); because of lack of adverse effects and blood pressure changes, it has been described as uroselective drug.

Androgen manipulation- the rationale for androgen suppression is based on that the embryonic development of prostate is dependent on DHT. 5ARIs (alpha reductase inhibitors) may be used to prevent progression of LUTS secondary to BPH and to reduce the risk of urinary retention. Dutasteride is a dual inhibitor of 5α reductase type 1 and 2, so greater impact on suppressing serum DHT. Other agents are antiandrogens, GnRH analogues, progestational agents have limited role in BPH. Combination therapy with α adrenergic blocker and 5α reductase inhibitor is usually preferred.

Phosphodiesterase inhibitors (PDEIs)- they act by smooth muscle relaxation in prostate, bladder neck or erectile tissue, increase oxygenation of bladder and prostate, has antiproliferative effect, decrease urgency. They improve urinary symptom score (level 1 evidence); especially for men with LUTS and significant ED. Sildenafil, Vardenafil, Tadalafil are some of the agents used. Headache, dyspepsia, flushing are some commonly observed side effects. Combination therapy with α blocker may lead to symptomatic hypotension and should be avoided

Case 2 :

67-year-male presented with acute urine retention which was relieved by per urethral catheterization. History of Poor urinary stream (5), Straining during micturition (4), Intermittency (2), Incomplete urinary bladder emptying (5), Frequency (5), Urgency (3), Nocturia (4); IPSS 28/35. Patient was a known case of diabetes for 20 years on inj insulin. He also had history of myocardial infarction 5 months back and on antiplatelet agents since then.There was history of bilateral inguinal hernia surgery 2 year back.

External genitalia – normal, Hernial sites – scars of previous surgery at bilateral inguinal region, no cough impulse. DRE- peri-anal skin – normal, anal tone – normal, prostate-grade 3, firm, smooth, non-nodular, non-tender, median sulcus not palpable, rectal mucosa freely mobile.

On further investigation ultrasound examination showed bilateral kidney mild raised cortical echogenicity without any hydroureteronephrosis, urinary bladder wall thickened, prostate – 92 cc with median lobe projection 1 cm, PVR- 163ml. No evidence of UTI and hematuria on urinalysis. Serum urea / creatinine was 38/1.3 and S. PSA- 3.9 ng/ml. Uroflowmetry showed – Voiding volume 160ml, Q max – 8 ml/sec.

Patient was diagnosed having BPH and started on medical therapy. Trial without catheter (TWOC) was given after 1 week and 4 weeks of á blocker therapy but patient could not void on both trial and hence put on PUC.

Considering the failed medical therapy and failed TWOC,the patient was planned for surgical intervention. Surgical options available for this patient were monopolar TURP (transurethral resection of prostate), bipolar TURP, LASER enucleation of prostate; of which we preferred for Thullium laser enucleation of prostate (THULEP) as procedure of choice, taking in account with continuation of antiplatelet agent.

Case 3:

A 59-year-male presented with bilateral pedal oedema, facial swelling for 3 weeks h/o overflow incontinence, increased frequency, nocturia, sensation

of incomplete evacuation of urine for 1 year.

History of Poor urinary stream (5), Straining during micturition (4), Intermittency (3), Incomplete urinary bladder emptying (5), Frequency (5), Urgency (4), Nocturia (5); IPSS 31/35.

Patient was a known case of hypertension on medications.Bilateral pedal oedema present.On abdominal examination bladder was palpable up-to the level of umbilicus, no other organomegaly. DRE- peri-anal skin – normal, anal tone – normal, prostate-grade 3, smooth, firm, non-nodular, non-tender, median sulcus not palpable, rectal mucosa freely mobile.

On further investigation ultrasound examination showed bilateral hydroureteronephrosis, urinary bladder wall thickened, prostate – 64 cc with median lobe projection 1 cm, PVR- 358 ml. Urinalysis showed plenty pus cells. Serum urea / creatinine was 67/2.3 and S. PSA- 7.9 ng/ml.

Patient was diagnosed having obstructive nephropathy. Patient was admitted and PUC was done. Post obstructive diuresis and decompression hematuria were looked for. Serial electrolyte monitoring and urine output measurement done. Patient had hyponatremia which was corrected as per protocol. S. PSA repeated after 3 weeks when UTI was ruled out on urinalysis,which was 1.89ng/ml. Repeat serum urea / creatinine was 45/1.8. As there was definitive indication of surgery in this patient, this patient was planned for monopolar TURP.

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